Correlation between Superoxide Dismutase and Chronic Periodontitis in Patients with Type II Diabetes

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ABSTRACT

The present study was designed to estimate and compare the superoxide dismutase (SOD) levels in the gingival crevicular fluid (GCF) and serum of type II diabetes mellitus patients and healthy individuals with and without periodontal disease. The study comprised 60 subjects, inclusive of both genders in the age group of 35 to 65 years. Patients were divided into two groups comprising 30 patients in each group. Patients were categorized into 30 chronic periodontitis patients with type II diabetes (test group) and 30 chronic periodontitis patients without diabetes (control group). The GCF and serum samples were collected and sent for biochemical analysis to estimate the SOD levels. Results obtained were then statistically analyzed using Student’s t-test. The SOD levels were found to be highest in the diabetic group. The serum SOD levels were found to be highest in the diabetic group. The increased levels of SOD seen in diabetic patients may be a result of a protective and adaptive mechanism against the oxidative stress developing in the tissue.

Keywords: Diabetes mellitus, Gingival crevicular fluid, Periodontitis, Serum, Superoxide dismutase.

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INTRODUCTION

Superoxide dismutase is an antioxidant enzyme that acts against superoxide, an oxygen radical, i.e., released in inflammatory pathways. It protects the cell against the deleterious effects of reactive oxygen species (ROS). Periodontitis is a term used to describe an inflammatory process initiated by a plaque biofilm formation that leads to loss of periodontal attachment to the root surface and adjacent alveolar bone. Diabetes mellitus is a metabolic disorder resulting from the defects in insulin secretion, insulin action, or both. The pathways of vascular tissue complications in diabetes are mediated through the formation of advanced glycation end products and increased production of ROS. The aims of this study are (1) to evaluate the level of SOD in subjects with chronic periodontitis with or without diabetes; (2) to assess the periodontal status in patients with chronic periodontitis diabetes; (3) to evaluate the correlation between the level of SOD in chronic periodontitis patients with and without type II diabetes.

MATERIALS AND METHODS

Sixty subjects were randomly selected from the Department of Periodontics, Jaipur Dental College, Jaipur, India, and the Department of General Medicine, Community Health Center, Amer. Informed consent was taken from all the subjects prior to enrolling them into the study.

Out of 60 patients, 30 chronic periodontitis patients with type II diabetes (test group) and 30 chronic periodontitis patients without diabetes (control group) were selected for the study.

Inclusion Criteria

- Patients should have minimum of 20 permanent teeth
- Patients with age ranging from 35 to 65 years in both sexes
- Patients who have not undergone any periodontal treatment in the past 6 months
- Patients diagnosed to have type II diabetes and who are on oral hypoglycemic agents for the diabetic group
- Patients diagnosed to have chronic generalized periodontitis with probing depth ≥4 mm and clinical attachment loss ≥4 mm

Exclusion Criteria

- Patients with any systemic disease or conditions apart from type II diabetes
- Patients who are on antibiotics/anti-inflammatory drugs/steroids in the past 3 months
- Subjects who had taken vitamins, mineral supplements, or antioxidants for the past 3 months
- Patients having history of any allergy
- Pregnant or lactating women
- Patients with chronic smoking and alcoholism

Source of support: Nil

Conflict of interest: None

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Patients were examined using a mouth mirror and UNC-15 probe. The periodontal status of the subjects was determined by measuring probing depth, clinical attachment level (CAL), gingival index given by Loe and Silness, and plaque index given by Silness and Loe. The subjects in both the groups were evaluated for glycated hemoglobin and SOD level.

**Gingival Crevicular Fluid Sampling**

In subjects, GCF was collected from teeth which have maximum pocket depth in the morning between 08:00 and 10:00 hours. The area was isolated with cotton rolls with attention to eliminate salivary contamination, and gently air dried. The paper points were inserted into the pockets until a slight resistance was felt and held in the sulci for 5 seconds with delicate care to avoid irritation of pocket or epithelium of sulcus (Fig. 1). Any paper point contaminated with blood was discarded and procedure was repeated at another point. Sample was kept in 1 mL Tris–HCl buffer (pH 6.5) and eluted for 30 minutes and stored at −4°C till analysis for SOD assay (Figs 2 and 3).

**Collection of Blood Sample**

Venous blood was collected from each subject in ethylenediaminetetraacetic acid-containing tubes (Fig. 4). One sample was used to analyze glycated hemoglobin. From another tube, plasma was prepared.

**Statistical Analysis**

Statistical evaluation of serum and GCF SOD levels was done using Student’s t-test. A p-value of less than 0.05 was considered to be significant.

**RESULTS**

**Serum SOD Levels**

The mean serum SOD levels of test and control were 32 ± 12.02 and 10.18 ± 3.57 respectively (Graph 1).
The SOD level in serum in test group was more when compared with control group, and it was statistically highly significant.

**Gingival Crevicular Fluid Superoxide Dismutase Levels**

The mean GCF SOD levels of test and control group were 32.90 ± 12.05 and 9.99 ± 3.57 respectively (Graph 2). The SOD level in GCF in test group was more when compared with control group and it was statistically highly significant (Table 1).

**DISCUSSION**

The present study was aimed to determine the SOD level in chronic periodontitis patients with type II diabetes (test group) and healthy patients with chronic periodontitis (control group). Results were in agreement with the studies done by Thomas et al, Rathod et al, and Baltacioglu et al. Increased SOD serum level in the diabetic group may appear as a result of a protective and adaptive mechanism developing in the tissue, and may also be an indicator of the increased generation of ROS in diabetes. Oxidative stress is the major reason for manifestation of periodontal disease and is caused due to an imbalance between harmful free radicals and protective antioxidants. A delicate balance exists between antioxidant defense and repair systems and pro-oxidant mechanisms of tissue destruction, which, if tipped in favor of tissue damage, could lead to significant attachment loss. Higher gingival-SOD activity in diabetes may be attributed to an adaptive mechanism in tissue 6 and tissue 21. Antioxidant benefits have been reported in diabetic patients treated with metformin. The mechanism was shown to be by a decrease in lipid peroxidation. This may explain the higher level of antioxidant concentration seen in diabetic patients with chronic periodontitis compared with chronic periodontitis patients.

**CONCLUSION**

Based on the statistical assessment of biochemical parameters, the following conclusion can be drawn from the study: (1) Serum SOD level was significantly high in diabetic group than in chronic periodontitis group; (2) GCF SOD level was significantly high in diabetic group than in chronic periodontitis group.

**REFERENCES**


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